337 Biomechanical Basis of Traumatic Brain Injury

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OVERVIEW

The complex pathophysiologic phenomena encountered in patients with traumatic brain injuries can be viewed, ultimately, as the brain's response to an external mechanical force. Preventing and treating the consequences of these injuries require an understanding of the mechanical factors that induce the injuries. In this chapter, we focus on the acute consequences of traumatic loading, referred to as the *primary injury*, including an overview of the principal mechanical forces that contribute to these brain injuries, how these mechanical forces cause movement and damage within the brain, and the predominant clinical consequences of the primary injury mechanism or mechanisms. Damage from the primary injury is often compounded over time by evolving and multifaceted pathophysiologic sequelae collectively referred to as *secondary injuries*, but that topic is beyond the scope of this chapter (see Chapter 346 and Loane and Faden[1] for a review). This chapter, which addresses the unique biomechanical causes and consequences of traumatic brain injury, is introductory; more detailed investigations of these principles can be found in other publications.[2,3]

CLINICAL CLASSIFICATION OF BRAIN INJURIES

Clinically, brain injuries can be classified into three distinct categories: skull fracture, focal injury, and diffuse brain injury. Although skull fracture requires significant biomechanical loading, it may or may not involve damage to the underlying brain, and, regardless, the fracture is often not a direct cause of neurological disability. Focal injuries are defined as visible damage to the parenchyma that is generally limited to a well-circumscribed region; examples of focal injuries include contusions to the cortex and subdural, epidural, and intracerebral hematomas. Focal injuries occur in nearly half of all patients with severe brain injuries and are responsible for approximately two thirds of brain injury–related deaths.[4] Diffuse brain injuries differ from focal brain injuries and skull fracture in that they often occur without producing macroscopic structural damage, are associated with widespread brain dysfunction, and appear in approximately 40% of patients with severe brain injuries.[5,6] Although contributing to nearly one third of deaths from brain injury, the most important aspect of diffuse brain injury is that this injury subtype is the most prevalent cause of disability in survivors of traumatic brain injury. In its mildest form (concussion), diffuse brain damage may not necessarily be structural and may involve only alterations in neural excitability, neurotransmission, or long-term changes in receptor dysfunction.[7] In more severe cases, diffuse brain injury manifests as prolonged coma without mass lesion and involves some degree of structural derangement at the microscopic level. Diffuse brain injury includes damage from both brain swelling and ischemic injury. However, the most commonly injured substrate in diffuse brain injury is the axons within the white matter; for this reason, the prominent form of diffuse brain injury is termed *diffuse axonal injury*.[4]

BIOMECHANICAL MECHANISMS OF INJURY

Types of Biomechanical Loading

Static or quasi-static loading is an uncommon occurrence and is used to describe force applied to the head very slowly, typically occurring over elapsed times longer than 200 milliseconds. These injuries generally involve a significant weight impinging on the skull for a prolonged time. Squeezing or crushing of the skull commonly occurs as a result of this static or quasi-static loading and probably involves fractures at the vault or basilar skull region. Remarkably, because the loading to cause this extensive fracture pattern is slowly applied, consciousness is commonly preserved after quasi-static loading. At high levels of compression, however, severe compression of the brain can lead to herniation of the brain contents and, often, fatal brain damage.

Dynamic loading is the more common type of mechanical loading to the head, especially in traumatic injury. Dynamic loading is applied rapidly, typically in durations of less than 50 milliseconds. Dynamic loading can be of three types: impulsive, impact, or blast overpressure. Despite the etiologic distinctions of dynamic loading, the fundamental means of damaging the skull and brain are the same: distorting or straining the bony or soft tissues beyond their functional or structural tolerance.

Impulsive loading occurs when the head is set into motion indirectly by a blow to another body region or by the sudden motion of another body region (e.g., torso). These conditions are common, inasmuch as a blow to the thorax or face often sets the head into violent motion and causes the brain to move within the skull; the interaction of the brain with the internal skull surface and intracranial membranes leads to injury along the brain surface and within the brain parenchyma. Sufficiently high levels of inertial forces to cause severe brain injuries are unlikely to occur without direct impact to the head or face, although this is theoretically possible. Thus when the head is set in motion by indirect impact to the chest, it is probably not possible to reproduce the entire spectrum of inertial injuries. However, lesser degrees of diffuse brain injury and cerebral concussion can be caused by impulsive loading.

Impact loading is the more frequent type of dynamic loading and commonly occurs as a result of motor vehicle accidents, falls, or sports collisions. Impact loading usually results from a combination of contact forces and inertial (acceleration) forces. The head response to impact conditions depends on the object that strikes the head. For example, inertial effects can be minimal if the head is prevented from moving when it is struck. In this situation, contact-related injuries occur both near and distant from the point of impact. Contact phenomena effects vary with the size of the impacting object, the magnitude of force delivered, and the force direction. Factors contributing to force magnitude include the mass, surface area, velocity, and hardness of the impacting object. For objects larger than approximately 2 square inches, localized skull bending occurs immediately beneath the impact point and peripheral to the impact sites. If the skull deformation exceeds the tolerance, skull fracture occurs. Penetration, perforation, or localized depressed skull fracture is more likely if the object has a surface area of less than 2 square inches. In addition, pressure waves can travel through the skull and parenchyma from the point of impact; within the brain, these pressure waves can cause localized stresses, distortion, and injury in the form of small hemorrhages.

Blast overpressure loading is a third type of dynamic loading that has received considerably more attention since the early 2000s because of the emergence of traumatic brain injury as the "signature injury" in the Iraq and Afghanistan conflicts. Sometimes referred to as *shock wave loading to the brain*, this loading is the delivery of a rapid-onset, very short (<5 milliseconds)

pressure wave to the brain that travels at the speed of sound within the tissue. The pressure wave may reflect at different interfaces in the brain (e.g., blood/tissue; cerebrospinal fluid/tissue) and cause microscopic damage at these interfaces. The relative movement of the head as the atmospheric shock wave passes across the head is almost unnoticeable, and therefore this type of loading can occur independently of either impulsive or impact loading. However, blast-induced injuries are often extremely complex as the passing of a "shock front" is often immediately followed by a powerful overpressure—sometimes referred to as the *blast wind* (a forced super-heated air flow)—that may load the subject impulsively (with possible impact deceleration upon hitting ground, wall, or other surface) or through direct impact with projected objects. With the recent focus on brain damage from blast overpressures, there is an emerging awareness that tolerance limits may need to be understood specifically for pressure loading, especially the multifaceted injuries that generally accompany blast exposure.

Tissue Properties and Responses to Loading

Across all these loading regimens, strain (or deformation) is considered the proximal cause of tissue damage. In general, strain can be considered as the amount of deformation the tissue undergoes as a result of applied mechanical force. Strain is often described as compressive, tensile, dilatational, or shear in nature (Fig. 337-1). Compressive strain is the amount of contraction caused when the material is compressed. For instance, if a stiff cylinder is placed upright on a tabletop and a stack of books is placed on the top circular face of the cylinder, the cylinder would shorten in relation to its original, unloaded length. If the cylinder was originally 10 cm in length and became 8 cm when the books were placed on top, the material is said to withstand a 20% compressive strain. In comparison, tensile strain is the amount of elongation that occurs when the material is stretched. If the column 10 cm in length becomes 11 cm long when stretched, it undergoes a 10% tensile strain (stretched length minus original length, divided by original length). Dilational strain, also referred to as *volumetric strain*, is the change in volume that occurs when pressure is applied to all exposed sides of a material. Most materials show negative or positive dilational strains when positive or negative pressures, respectively, are applied to the material. Shear strain can be considered the amount of distortion that occurs in response to forces applied all along the surface of the material. A common illustration of shear strain is the distortional change that occurs in a neatly stacked deck of playing cards when one hand is moved across the top of the deck. None of the cards are compressed or stretched as a result of this motion, but the side profile of the deck changes from a rectangle to a parallelogram. The amount that the side profile varies from a normal rectangle indicates the state of shear strain.

The strain limit of bony and soft tissue before damage occurs depends not only on the force (e.g., direction, magnitude, duration) but also on the mechanical properties of the tissue. Materials such as concrete are ideal for sustaining large compressive loads, but they need reinforcement to sustain the same loads or deformation under tension. For biologic materials, bone is a prototype material that exhibits high compressive strength and is weaker in tension and shear. In comparison, rubber materials can often reach deformations two or three times their original length before breaking. Brain tissue is one of the softest materials in the body, and its mechanical behavior is often based on descriptions used for rubber-like materials, easily reaching deformations of 20% to 50% before failing.

In addition to the relative stiffness of different living tissues, biologic materials often show a stiffening when the rate of applied force increases; that is, dynamic loads cause less deformation than do the same forces applied more slowly. Materials that show a change in stiffness with applied loading rate are termed *viscoelastic*. Perhaps the most recognizable viscoelastic material is Silly Putty (Crayola LLC, Easton, PA). Silly Putty can be easily formed into various shapes by hand. If pulled slowly, Silly Putty can lengthen and deform substantially before breaking. If, however, this material is pulled very quickly, the material breaks at a much shorter length. Biologic tissues typically display such viscoelastic behavior and can therefore withstand strain better if they are deformed slowly, rather than quickly.

In addition to the material properties of the tissue, the mechanical tolerances of these tissues—which can be thought of as thresholds for irreversible damage—are the critical determinant in the conditions that cause injury. The three principal

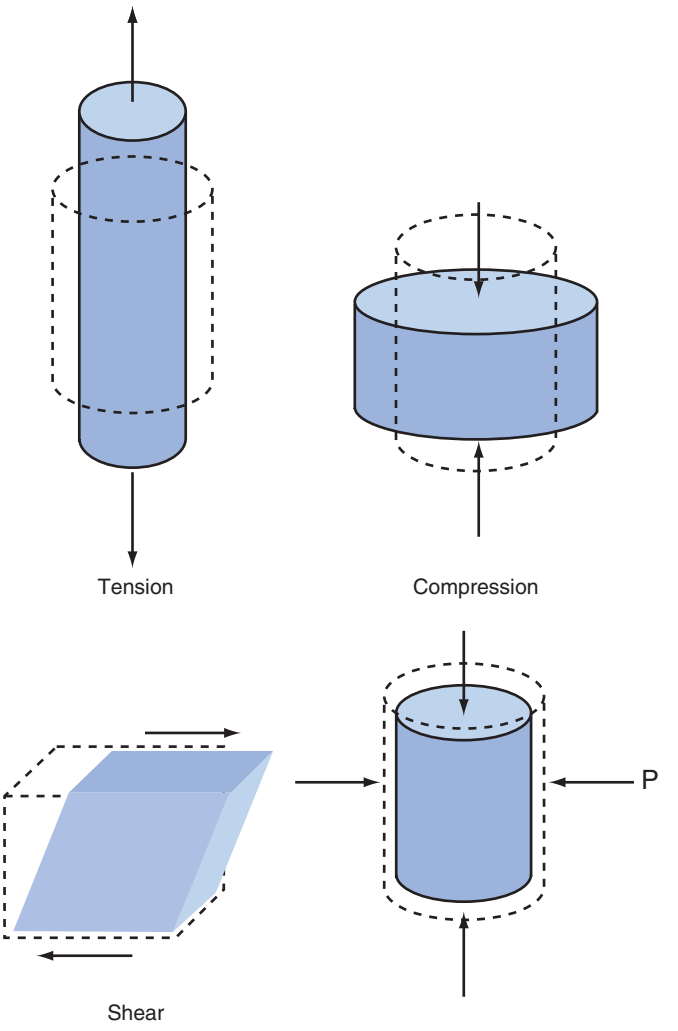

Figure 337-1. Types of strain (deformation). Illustration of the different types of strain associated with traumatic intracranial damage. The *dashed lines* represent the undeformed shapes; the *shaded objects* represent the deformed configurations. Tearing of vessels is often described in terms of a tensile strain limit, in which the limit is described as a fraction (usually percentage) of the original vessel length. Bone tissue can sustain both tensile and compressive loading, and the failure limit for each type of loading is normally distinct. Soft tissues, notably brain tissue, are susceptible to shearing deformation. Axons within the tissue undergo more complex deformation. Pressure (P) applied to all sides of a material causes a dilational (volumetric) strain, which may be a mechanism to consider at very high loading rates (e.g., blast-induced pressure waves). *Arrows* in each diagram indicate the force direction that causes the resulting deformation.

tissues involved in brain injury (bone, vascular tissue, and brain tissue) vary considerably in their tolerances to compression, tension, and shear. Bone, for example, is considerably stronger than either vascular or brain tissue; substantially more force is needed to induce damaging levels of stress. The amount of strain that bone can tolerate is actually less than that needed to injure brain tissue (e.g., bone breaks at 1% to 2% strain, whereas brain and vascular tissue may not tear until 10% or 20% strain is applied). The key difference is the stiff mechanical properties of bone in comparison with either brain or vascular tissue: it takes considerable force to cause 1% to 2% strain in bone. Like vascular and brain tissue, bone also withstands compressive strain and shear strain, with a tensile strength tolerance somewhere in between. There is proportionately less difference among the three strain tolerances for bone, whereas there is a considerable difference in the tolerance of brain tissue to tension, shear, and compression.

Because brain tissue is virtually incompressible in vivo and it has a very low tolerance to tensile and shear strain, both tensile and shear strains are usually the causes of brain damage. The same is true for vascular tissue. Whether vascular or brain tissue damage occurs depends on the exact properties and tolerances of these two tissue types. As discussed later, vascular tissue tends to fail under more rapidly applied loads than does brain tissue and, depending on the type of loading to the head, under certain conditions, relatively pure injury can occur with vascular elements rather than the neural elements within the head.

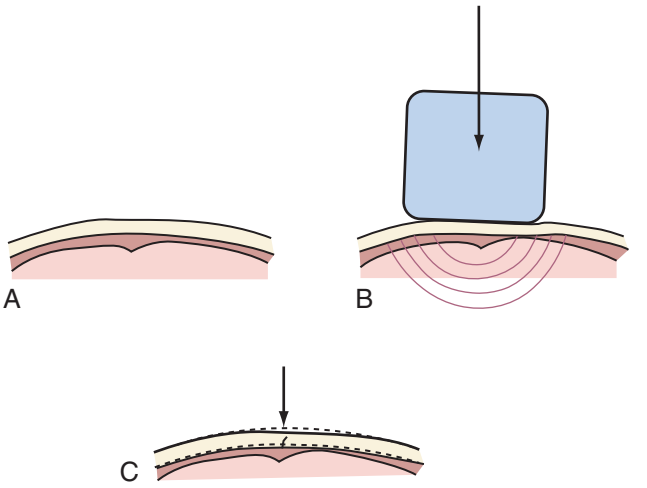

Figure 337-2. Local contact effects. Simple schematics of skull and underlying brain tissue **(A-C).** During an impact (*arrow* in **B**), the scalp and underlying skull undergo deformation. With its stiff mechanical properties, bone bends inward, which creates localized stresses within the bone and adjacent brain tissue (stress waves indicated as *rounded lines* emanating from the impact site in **B**). The stress within the bone is tensile on the inner table and compressive along the outer table. Most bony structures fail under tension before compression, and therefore the initial site of skull fracture occurs along the inner table (**C,** *arrow*).

MECHANISTIC CAUSES OF HEAD INJURIES

Most head injuries are caused by one of two basic mechanisms: *contact* or *inertial* loading. In contact injuries, the head strikes an object or is struck, regardless of whether the blow causes the head to move thereafter. Inertial injuries are often called *head motion* or *acceleration* injuries because they result from violent head motion, regardless of whether the head moves because of a direct blow. Although contact or inertial injuries may happen in isolation, it is most common for these injury mechanisms to occur in combination (e.g., contact loading that results in sudden head acceleration). In addition, shock wave propagation associated with blast-induced traumatic brain injury may represent a third basic mechanism of injury, although it is not as well understood.

Contact Injuries

In general, contact injuries are caused by force during impact. These injuries result solely from contact phenomena and are not caused by head motion or acceleration. Pure contact injuries can therefore be considered trauma that would occur if the head were prevented from moving: for instance, if the head was impacted by an external object while the subject's head was held fixed against a wall. In this scenario, the contact injury would be the skull fracture and underlying contusion that occurred from the impact force.

Because most impacts also cause head motion to some degree, contact injuries rarely occur alone clinically. Rather, acceleration injuries are superimposed on contact injuries. Contact forces are of two types: local effects at or near the impact site and effects remote from the area of impact. In both instances, contact forces cause only focal injuries; they do not cause diffuse brain injury.

Local Contact Effects

Examples of injuries from local contact effects include most linear and depressed skull fractures, some basilar skull fractures, epidural hematomas, and coup contusions. These injuries commonly occur when an object moving at a high velocity strikes the head: for instance, a baseball or a baseball bat. Penetrating injuries by foreign objects, such as a bullet or shrapnel, also fit in this category. A linear skull fracture results from skull bending that occurs locally at the impact site and exceeds the local strain limit for the bony tissue (Fig. 337-2).

Strain tolerance is related to inherent mechanical properties of the material; therefore, it is not surprising to find that skull fracture depends partly on the material properties of the skull and its thickness in the region of impact. Additional factors include the magnitude and direction of impact and the size of the impacted area. Mechanistically, the local inward bending caused by the impact creates compressive strains in the outer skull surface and tensile strains on the inner surface (see Fig. 337-2). Bone, naturally resistant to compressive forces and strains, is less resistant to the tensile forces on the inner skull surface. Thus the initial fracture begins at the inner table. Once initiated, the fracture follows a path of least resistance that is dictated by the geometric and strength characteristics of the surrounding bone of the skull. During the continuing fracture process, energy from the impacting object is transferred to the skull via the fracture. The linear fracture is complete when the impact energy is dissipated completely.

Depressed skull fractures occur when the striking or struck object is small enough to cause concentration of strain and stress immediately beneath the impacting object. These concentrated strains produce a highly localized fracture pattern that does not emanate from the contact site. Unlike a linear skull fracture, energy is not absorbed by a fracture propagating away from the fracture point. Instead, the energy is dissipated by the localized bone failure. For highly concentrated contact forces, these depressed skull fractures penetrate completely through the skull to damage the underlying tissue. Impact to the skull base or nearby regions can occur and cause basilar skull fractures from local contact effects. Direct impacts to the occiput or mastoid are common mechanisms of this skull fracture type.

Vascular damage caused by local contact effects (e.g., epidural hematoma and coup contusions), is often intimately linked to

skull fracture types. The epidural hematoma results from a complicated form of skull bending, generally from a fracture. In this case, dural vessels are torn as the fracture propagates and travels past a vessel. The mechanical failure of these vessels can also occur without fracture, inasmuch as skull deformation and bending may be sufficient to cause vascular tears.

Coup contusions occur beneath the site of impact under certain conditions. These contusions are caused by direct injury to the brain and the surface vessels that lie beneath the area of skull deformation or by the high negative pressures that develop in the area where the skull rapidly snaps back into place. The first mechanism causes highly focused compressive strains; the second subjects the brain to very high tensile stresses. In either case, the strains are sufficient to cause tissue failure of the pial and cortical vessels of the brain and form localized contusions. Brain laceration is an extension of the same phenomenon but may also occur if inward bending of the skull is sufficient to actually perforate the pia.

Remote Contact Effects

Contact phenomena may also result in remote injuries because of complex skull distortion or stress wave propagation, potentially culminating in skull fractures away from the injury site and contrecoup brain contusions. Remote vault fracture can develop if the impact occurs over a thick portion of the skull or if the striking object is relatively broad. Because the thick skull can withstand the impact force, the local inward-bending energy can travel away from the impact site to affect remote skull regions, which may then undergo larger local bending because of their inherently weaker characteristics. If the strain tolerance is exceeded, remote skull fracture occurs. This raises the possibility that an impact at one site could lead to fracture distant from the impact site. Once initiated, a fracture usually propagates along the lines of least resistance. Typically, regions such as the basilar skull area have thin skull sections that offer this path of least resistance. As a result, various types of basilar skull fractures may occur from remote contact loading.

On occasion, head contact is severe enough to cause global changes in skull shape. These global changes are particularly apparent if the physical skull structure is compliant, as in infants and developing children. This type of large skull deformation can cause rapid increases or decreases in intracranial volume. These changes are usually transient and, because of the elastic nature of the skull and its contents, the skull generally returns to its normal shape immediately after the force is removed. Two phenomena that may appear at these large deformations are localized pressure changes and intracranial volume fluctuations, and they cause a variety of injuries. The rapid changes in skull shape can be sufficient to produce levels of negative pressure at points where the skull has pulled away from the brain and caused contrecoup contusions. In a similar injury, the separation of the brain surface from the dura mater can also lead to the rupture of surface vessels and subdural bleeding. This localized pressure mechanism is proposed as a cause of small petechiae surrounding the ventricles, presumably as they expand in response to brief negative intracranial pressures. A sudden fluctuation or decrease in intracranial volume caused by global skull deformation can prompt herniation of the brain contents through the various foramina, primarily the foramen magnum. The action of herniation places an excessive amount of strain on structures for the lower brainstem; thus it can potentially cause maximal injury at tissue remote from the impact site. These intracranial volume fluctuations may explain part of the distinct neurological and pathologic observations found in infants or children with traumatic brain injury. However, the frequency with which trauma contributes to global skull deformation in the adult is still debated, and these injuries are probably much more commonly results of inertial effects.

The second mechanism for remote damage from contact loading is stress waves originating at the point of impact. Radiating in a three-dimensional manner from the loading point at an rapid speed, stress waves spread through the skull to cause local skull distortions that, if excessive, produce basilar and remote vault fractures (as discussed previously). However, stress waves also spread throughout the brain and, like waves in water, reflect from the opposite side of the head and reverberate within the brain. How these waves reverberate within the brain depends on, among other factors, the ability of the brain tissue to dissipate the disturbances at the impact site. If the stress waves in the brain are amplified by this reverberation or local skull bending, high-intensity localized pressurized differences occur. If the strains induced by these stress waves exceed the tissue/vessel tolerance, damage results. In theory, the areas of stress concentration caused by reverberating pressure waves occur deep within the brain, not at its surface. Therefore, pressure waves have been used to explain the formation of the intermediate coup contusions (a term sometimes used to describe hemorrhages occurring on nonconvex surfaces), scattered deep intracranial hemorrhages, and traumatic intracerebral hematomas. However, because these waves travel so rapidly and are quickly dissipated, this mechanism remains a matter of debate.

Head Motion (Inertial) Injuries

Inertial loading of the head, whether from impact or from impulsive loading, causes a broad variety of clinically relevant injuries. These are commonly called *acceleration* and *deceleration injuries* because acceleration is an important physical measure for loading. Other factors (e.g., head velocity) may be equally important, and although this category should perhaps be called *head motion injuries*, the term *acceleration injuries* remains in use. From the mechanical point of view, acceleration and deceleration are the same physical phenomenon and differ only in direction. For example, the effects of accelerating the head in the sagittal plane in the direction of posterior to anterior are the same as those of decelerating a head from anterior to posterior.

Like to contact injuries, head motion results in strains within the brain tissue that can cause either functional or structural damage (Fig. 337-3). First, *differential movement of the skull and*

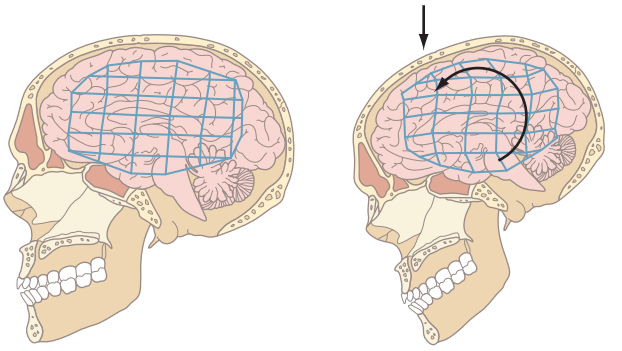

Figure 337-3. Head motion injuries. Impact can cause local contact effects, but two additional effects contribute to the lesions observed clinically: because of its inertial properties, the brain slides in relation to the inner skull surface *(circular arrow),* and cortical vessels connecting the brain to the dural membrane may tear. In addition, the inertial loading delivered to the brain, coupled with its soft material properties, leads to a deformation of the brain contents. This deformation is visualized with a superimposed grid pattern, which undergoes deformation from the acceleration imparted during impact (represented by *straight arrow*).

brain can be produced by head acceleration or motion. This relative movement occurs because the brain is free to move to some degree within the skull and because, as a result of inertia, the brain lags behind for a brief moment after acceleration begins. When combined, these factors allow the skull and dura to move in relation to the brain surface, which potentially causes localized strains at the surface. Particularly susceptible in this situation are the parasagittal bridging veins between the brain surface and dura, which may tear if the strain exceeds the vascular tolerance. Furthermore, the movement of the brain away from the skull creates a region of low pressure that, if sufficiently intense, causes contrecoup contusions. Second, head acceleration can produce *strain within the brain parenchyma* and therefore can cause widespread disturbances to brain function or structures. Strain within the brain parenchyma can manifest as classic cerebral concussion, diffuse axonal injury and associated tissue tear hemorrhages, and most intermediate coup contusions. In each type of injury, the severity and extent of damage are intimately linked to the magnitude, rate, duration, direction, and type of inertial loading.

Types of Head Acceleration

Three types of acceleration can occur: translational, rotational, and angular (Fig. 337-4). *Translational acceleration* occurs when the center of gravity of the brain (which is approximately in the pineal region) moves in a straight line in any direction but the head is not able to rotate. Translational acceleration alone is uncommon because physiologic articulation of the head and neck limit the purity of this movement. Exceptions occur when the head moves in a translational manner for brief time periods, or the head can become arrested with other motions. An exception may be vertex impact, during which superior-to-inferior motions can occur. The brain motions that take place during translational acceleration result primarily from the relative brain-skull motion previously described and not from strain produced deep within the brain. Concussive injuries do not occur when the head experiences a purely translational acceleration.[8] For concussion or diffuse axonal injury to develop, the brain must undergo angular acceleration. Therefore, translational acceleration does not cause diffuse brain injuries, but it can produce focal injuries, including contrecoup contusions and intracerebral and subdural hematomas.

Rotational acceleration occurs when there is rotation about the center of gravity of the brain, without the center of gravity itself moving. Because the center of gravity of the brain is in the pineal area, pure rotational acceleration is a virtual impossibility in nearly all clinical situations. For the brain to rotate around an axis that goes through the pineal area, the entire body would have to swing around it. A notable exception is when the head is rotated solely in the horizontal plane, where a pure rotation may occur about an imaginary vertical axis running through the pineal area.

Rotational acceleration is a very important and highly injurious mechanism because it not only produces not only the high surface strains seen in translational motions but also, because of the mechanical properties of brain tissue, it is also the only mechanism capable of producing high levels of strain deep within the brain itself. However, because of the infrequency of pure rotational motions in clinical situations, the effects of rotational acceleration are usually seen after angular acceleration of the head.

Angular accelerations occur when components of translational and rotational accelerations are combined. In this situation, the center of gravity moves in an angular manner. Because of the neck anatomy, angular acceleration is the most common head motion encountered clinically. Frequently, the center of rotation occurs in the lower cervical region. The exact location of this rotation point, in combination with the impact force magnitude, determines the proportion of translation and rotation that the brain undergoes. As the rotation point moves higher up the cervical spine, the rotational component is proportionally greater; moving the rotation point lower introduces proportionally more translational acceleration. As might be expected, angular acceleration is the most damaging mechanism of brain injury because it combines the injurious mechanisms of both translational and rotational movements, especially the latter. Virtually every known type of head injury can be produced by angular acceleration, except for skull fracture and epidural hematoma.

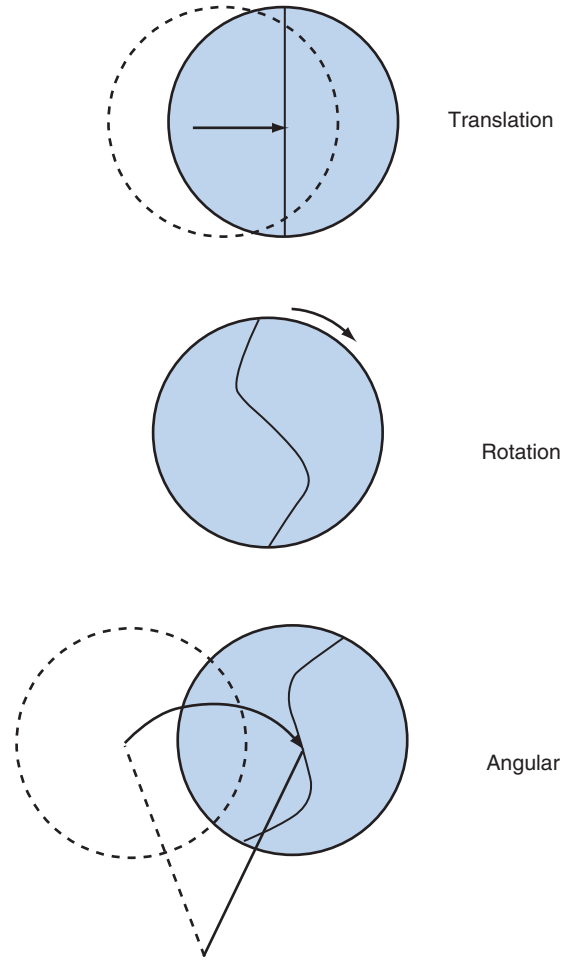

Figure 337-4. Types of head acceleration. Translational acceleration moves the head in a linear path. Alternatively, rotational acceleration induces a rotation about the head's center of mass, located approximately in the pineal region. Instances of pure translation or rotational accelerations are rare in real-world injury situations; rather, an angular motion is more common. The angular motion leads to a combination of translational and rotational acceleration, thereby creating injury patterns that arise from both acceleration types. The *dashed circles* represent the original position of the head; the *arrows* indicate the direction of impact; and in bottom illustration, the angle of motion is represented by the *solid* and *dashed lines*.

Determinants of Acceleration Injury

Several simple descriptors of acceleration—the amount and type of acceleration, its duration, and the rate at which acceleration is applied to the head—are interrelated and together contribute to the pattern of injury observed within each individual. Certainly, these parameters are linked; for example, for a constant level of

acceleration, as duration increases, head velocity and movement also increase. In past efforts to describe the tolerance of the head to different forms of traumatic brain injury, researchers have used two primary measures—(1) acceleration and (2) head velocity/duration of acceleration—to reflect this complex relationship between motion and resulting injury. Structural damage to superficial vascular tissue, especially to bridging veins and pial vessels, occurs in high-acceleration, short-duration conditions, whereas damage to brain tissue occurs in circumstances of high accelerations with longer pulse durations and thus higher velocities (as described in more detail later in this chapter).

The amount of inertially induced damage depends not only on the type of acceleration of the head but also on several other factors. Acceleration magnitude can be viewed as proportional to the amount of strain delivered to the brain, and acceleration rate is proportional to the strain rate. Both the strain and strain rate are factors contributing to the structural or functional limit of intracranial tissues. If the acceleration magnitude is constant, the rate of acceleration varies inversely with the duration over which the acceleration occurs. Conversely, if the acceleration duration is constant, acceleration rate varies directly with the acceleration magnitude.

When the amount of acceleration is constant and only the duration over which the acceleration occurs varies, three zones of clinical interest are encountered (Fig. 337-5). First, in very brief accelerations, many of the inertial effects within the brain are damped and, as a result, the brain actually experiences very little strain. As a result, extremely high accelerations are required to produce injury. Second, if the acceleration duration is slightly longer, strains begin to appear within the brain but are restricted primarily to the periphery. Moreover, the brain surface can slide in relation to the skull/dura surface. Injuries produced in these circumstances are confined to the brain periphery and vessels (e.g., bridging veins). Third, as the duration of acceleration lengthens even more, strains propagate deeper within the brain and can cause diffuse axonal injury that, in a severe form, manifests as prolonged traumatic coma.

In the first zone described, the strain rates are already so high that increasing acceleration adds little to the pattern of injury. In the second zone of injury, vascular tissues of the brain surface are clearly jeopardized; therefore, increasing the strain rate further by increasing head acceleration levels causes disruption of a higher number of vessels along the brain surface. In the third zone, in which the strain rate produces damage to brain tissue but is insufficient to produce damage to vessels, increasing the acceleration magnitude can increase the strain sufficiently to cause vascular injury. The results can be a combination of diffuse axonal damage with tissue tear hemorrhages, subdural hematoma, or both. These examples highlight the complexity of mapping acceleration profiles into exact patterns of damage within the brain.

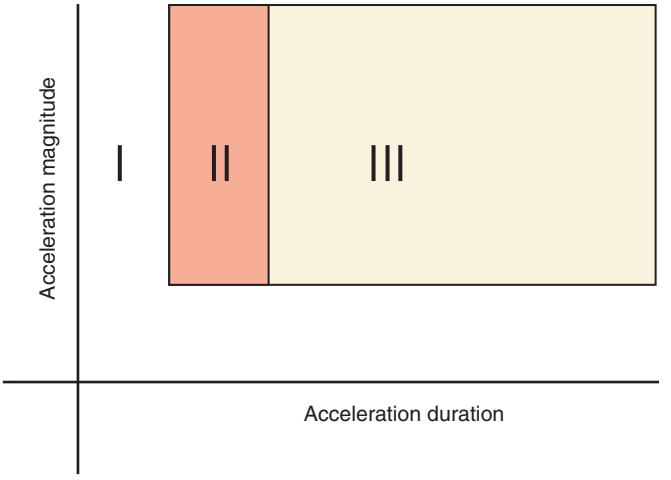

Figure 337-5. Zones of clinical injury. In an abbreviated format to describe the human tolerance to specific head motions, both the magnitude and duration of acceleration are described. A range of both parameters is associated with either injury or no injury. For rotational motions, there are three general zones. In zone I, very brief durations of acceleration are highly unlikely to cause injury, inasmuch as the strains do not propagate to deeper brain structures and the relative motion between the brain and skull is small. In zone II, slightly longer acceleration durations allow strains to penetrate the periphery but do not move significantly into the deep brain tissue. Therefore, injuries to the brain periphery and cortical vessels are more common. In zone III, even longer durations of acceleration enable the strains to propagate throughout the brain, and both the periphery and deep brain tissue can be injured.

Blast-Induced Brain Injuries

Awareness of the effects of blast loading on humans originated during World War I; the term *shell shock* was used to describe a collection of symptoms that included temporary altered mental state or confusion immediately after the blast.[9] In more severe forms, unconsciousness would occur. Although it was periodically addressed in the literature as a clinical syndrome, authors reporting more recent studies have better described the conditions of blast-induced brain injury.[10-12] Although blast-induced injury is formally defined in four phases, the bulk of blast-induced traumatic brain injuries occur in the primary, secondary, and tertiary phases. The primary injury phase comprises the response of brain tissue to the blast wave (the intense overpressurization impulse component of blast). The secondary injury phase results from focal impact or shrapnel penetration of the head. In the tertiary phase, injury results from head acceleration/contact as the body is moved by the "blast wind."

No one model can mimic the clinical and mechanical complexity of a blast-induced traumatic brain injury. As described previously, for injuries in the civilian population, the mechanical factors are often grouped into either the focal/contact or acceleration/inertial forces that produce the different mixture of clinical injuries ranging from concussion to skull fracture, contusions, and diffuse axonal injury. However, blast-induced injuries as a group result from the different physical aspects of the blast phenomenon; in short, primary blast-induced injury is from the shock wave, secondary blast-induced injury is from shrapnel (focal), tertiary blast-induced injury is from the "blast wind" (head acceleration), and quaternary blast-induced injury covers any remaining mitigating factors (e.g., thermal, chemical). These groupings apply not only to studying brain injuries, but also to other body regions susceptible to injury from blast. This distinct method of grouping blast-induced injuries may seem inconsistent with the classifications often used for civilian traumatic brain injury and is largely a result of having to reconcile two independently developed classification systems. However, a fair level of knowledge obtained from civilian traumatic brain injuries—most commonly resulting from falls, assaults, and motor vehicle accidents—can be used to study the implications of blast exposure for military personnel.

The primary phase of blast-induced injury is limited to injuries caused by the rapidly expanding blast wave.[13] In past work, researchers identified typical pressure wave profiles generated during blast events[14] and described how this pressure wave propagates through air and biologic tissues.[15-17] A shock wave from a blast is typically characterized as a rapid rise and fall of high pressures.[18] The shock wave passes in milliseconds, although the

exact profile and range are dependent on the size, type, and shape of the explosive. In air, the shock wave oscillates between overpressure and underpressure segments, but these waves dampen out quickly. In water, the shock wave maintains a normal pressure profile over a longer range. The presence of structures and fluid interfaces complicate this pressure profile.

Maximal stresses, and hence most structural damage in the body, occur when the shock wave travels from water to air. For this reason, a large number of studies focused on the fluid-air interfaces in the body, which are highly vulnerable to the passing pressure waves. Studies in these areas have generated injury thresholds for both the pulmonary system and the gut and bowels.[19] As a pressure wave travels through the organs, stress concentrations arise at tissue-air interfaces, stress waves are reflected at tissue interfaces, and possible constructive interference of stress waves within internal points of tissue can further complicate the pattern of injury.[18] Models to study the primary injury phase should account for both the blast pressure transmitted through the organ of interest and shock wave reflection/transmission behavior at tissue interfaces. More subtle changes in the mechanical properties of deep structures within the brain (e.g., blood vessels, cerebrospinal fluid, gray matter, white matter) may also contribute to interfacial stresses as the overpressure propagates through the brain, but more experimental evidence is needed to support this assertion.

Secondary blast-induced injury covers both the penetrating and nonpenetrating injuries that occur when high-velocity projectiles/fragments hit the head.[13] These injuries share some common characteristics with ballistic wounds in civilians but are considerably more complex because of the nonuniform size and number of the impacting fragments. Nevertheless, the mechanics of these penetrating lesions are becoming better understood, given a renewed emphasis on gunshot wounds in the civilian population. A projectile moving through soft tissue at high speed causes rapid expansion and subsequent collapse of tissue along the penetration tract. This induces cavitation damage along the path of the projectile, as well as primary laceration damage along the path of the fragment. At the mechanistic level, these injuries are best modeled as a tissue laceration, with the obvious complicating mechanisms related to blood in the extracellular space, and have the potential to cause secondary brain injuries, such as hypoxia.

Tertiary blast-induced injury includes the effects when the primary "blast wind" causes the subject to be thrown (i.e., rapidly accelerated) and subsequently hit fixed or mobile objects. These types of injuries share the most in common with contact/acceleration injuries in civilians, in which both the contact and acceleration forces can contribute to the different intracranial injuries that appear. Helmets worn by soldiers reduce the likelihood of injuries from direct contact for most of the head. However, current helmets are not designed to specifically reduce rotational acceleration after a impact to the helmet.[20,21] Thus there remains a high potential for inertially based injuries that include subdural hematoma and diffuse axonal injury. As a result, the predominant mechanisms for tertiary blast-induced injuries in helmeted soldiers are the intracranial deformations caused by the head's striking an object (i.e., contact injury) and the head's being struck by an object with sufficient mass to cause a significant inertial load (i.e., angular acceleration injury). In nonhelmeted victims, tertiary blast-induced injuries can also include skull fracture or contusions from the focal contact forces when the unprotected head strikes an object or surface.

PREDOMINANT CLINICAL CONSEQUENCES OF INJURY MECHANISMS

The clinical classification system for head injuries described at the beginning of this chapter is used to distinguish individual cases. It is clear that many of these injuries may occur in combination, and isolated occurrences of these injuries, particularly severe head injuries, are rare. Indeed, these injuries usually occur in complex accident situations, which underscores the multifaceted nature of most clinical cases. Nonetheless, certain particular head injuries are associated primarily with motor vehicle accidents, and other injuries occur more frequently with falls, with assaults, and in pedestrian accidents.

Skull Fracture

Linear Fracture

Linear fractures may occur solely because of the contact effects of impact. Head motion and acceleration play no role in the genesis of fracture. Impact by a hard object can cause linear fracture, in which most of the energy from the subject deforms the skull locally. Little energy is used to move or accelerate the skull. To prevent a more localized depressed skull fracture, the impacting object should generally be larger than approximately 2 square inches. However, if the impacting object or surface is considerably larger, then contact is distributed across a broad area of the scalp, and local bending effects are not present. Acceleration injuries may occur in parallel with an impact that will cause fracture, inasmuch as most impact situations set the head in motion and, therefore, cause a head acceleration that is superimposed on the contact loading effects. Therefore, skull fracture is commonly observed in combination with other types of brain injuries.

Depressed Fracture

Impact of small, hard objects causes depressed fractures. In this case, energy dissipation from the impact results in skull fracture immediately beneath impacting object. If the impacting force is substantial, all bone under the site of impact is damaged, and skull perforation occurs. Because of the focused loading, little or no propagation of the fracture occurs.

Basilar Fracture

A result chiefly of either direct impact or the propagation of stress waves through the skull as a result of the remote impact, basilar fracture may also occur as a consequence of the impact to facial bones. The thin basilar skull is particularly susceptible to remote contact effects because the structure of this region is considerably weaker and not as effective in managing local skull deformations initiated by remote impact. Common impact points for producing basilar skull fracture include the skull base, facial or mandibular bones, and remote skull impact points.

Focal Brain Injury

Epidural Hematoma

Epidural hematoma is generally a complication of linear skull fracture, although epidermal hematoma may occur without bone fracture. Typically, during the fracture initiation or propagation period, vessels in the underlying dural membrane are torn and bleeding ensues in the epidural space. These hemorrhages are generally arterial and, as such, may have grave consequences, including pressure effects, tissue dislocation, and herniation effects. Alternatively, the local skull bending caused by an impact may be sufficient to tear dural vessels without exceeding the failure limit of the bone, which would result in subdural bleeding without skull fracture. Epidermal hematoma is an impact-based phenomenon; therefore, no head motion or inertial effects cause an epidural hematoma.

Coup Contusions

Immediately under the impact point, coup contusions arise principally from the local bending or fracture of the skull caused by an impact from a relatively small, hard object. These phenomena, in turn, subject the underlying cortical and pial vascular network to strains that, if excessive, cause bleeding at or near the brain surface. Damage is likely to occur when the skull is "rebounding" from the impact and the vessels are experiencing tensile deformations. For cases of skull fracture, the localized strains from the skull's contacting the cortical surface provide a means for the vascular damage, noted in the pia and cortex.

Contrecoup Contusions

Although global skull deformation caused by impact may create tensile stresses and contusion damage remote from the site of impact, the predominant mechanism for contrecoup contusions is believed to be rotational acceleration. Indeed, two phenomena have historically been attributed to the pathogenesis of countercoup contusions: cavitation effects and inertial loading. Of the two, the more likely mechanism of the contrecoup damage is inertial: translational or angular head motion. The theory of cavitation effects is based on the idea that on impact, the brain moves toward the impact site, and an area of negative pressure develops directly opposite the point of loading. This negative pressure may, in turn, cause damage by exceeding the tensile strength of neural tissue or, alternatively, by causing small gas bubbles to appear within the parenchyma. The return to normal or positive pressures could then cause the small bubbles to collapse; this is termed *cavitation*. However, it is difficult to conduct experiments that clearly support the presence of cavitation as an injury mechanism, and this mechanism does not easily explain contusions located outside the region considered opposite the impact site. Similarly, the propagation of stress waves through the brain from blast overpressure may also cause areas of brief negative pressure and cavitation, although direct experimental evidence at survivable overpressure levels is necessary to support the relevance of this mechanism in blast-induced traumatic brain injury. Instead, it appears vascular disruption and cortical damage in contrecoup regions are primarily acceleration effects resulting from either translation or angular head motions. Each head motion, particularly angular movement, is capable of producing shear and tensile strains throughout the brain. If the strain fields are greater than the vascular tolerance in a given region, contusion occurs. However, impact is not necessary for contrecoup contusions to occur, inasmuch as these associated strain patterns generated by inertial forces can be caused by impulsive loading alone. The term *contrecoup* may be somewhat misleading: the critical mechanism is most often acceleration and not directly the contact effects from impact. Indeed, in situations in which the head undergoes impulsive loading, contrecoup contusions occur solely because of the strains generated in the cortical brain region during the acceleration period. Strain concentrations may occur in specific regions of the brain as a result of geometric effects and are responsible, in part, for the high incidence of frontal and temporal lobe contusions observed clinically.

Because of their macroscopic and easily identifiable nature, contusions are periodically used to characterize the biomechanical input to the head. However, several points must be mentioned when contusions are used as a tool in this manner. First, the line of action of an impact force cannot be ascertained simply by connecting a line between the coup and contrecoup damage sites. The discussions in the preceding paragraphs highlight the significance of inertial loading in producing the pattern of contrecoup damage, in comparison to the role of local contact effects. Thus coup and contrecoup injuries, although differing only slightly in name, arise from fundamentally different mechanisms. Contrecoup contusions are often not exactly opposite the point of impact. In fact, contusions of the temporal and frontal poles are contrecoup in almost every instance, regardless of the impact site. In consideration of these differences, it is more appropriate to consider a contrecoup contusion as one that is simply not immediately below the impact site; it should not be considered as appearing immediately opposite the point of impact. Second, in a similar manner, coup and contrecoup contusions should not be viewed as arising from acceleration and deceleration of the head, respectively. Rather, the relative proportion of coup versus contrecoup contusions depends solely on the response of the head to loading. Because of the apparent pathogenesis, the acceleration of the head caused by a concentrated blow has led to the proposal that acceleration causes coup contusions. However, a hard, small object that causes impact in these typical cases tends to produce focal skull deformation with underlying coup contusion, and a large portion of the energy is dissipated at the impact site. In these cases, local skull deformation produces a noticeable coup contusion, and the lack of substantial head motion produces slight or very limited contrecoup contusion. On the other hand, a softer or larger impact object commonly causes a deceleration injury, which causes less local injury beneath the point of impact, inasmuch as a significant proportion of the loading energy is used to set the head into motion or stopping it from moving. In this case, a large contrecoup injury may occur with a coup contusion that is smaller or without a coup contusion.

Intermediate Coup Contusions

Intermediate coup contusions are vascular disruptions of brain surfaces that are not adjacent to the skull. Although the mechanism of these lesions has not been studied extensively, it is likely that they are due to strain concentrations resulting from impact-generated stress waves or from inertially generated brain movements. In the latter case, the brain is thought to strike or be pulled away from the adjacent structures, which results in compressive or tensile strains, respectively. Alternatively, intermediate contusions of the cingulate gyrus can be caused by interactions with the falx, and, similarly, those of the inferomedial temporal lobe can result from involvement with the tentorium of the petrous ridge.

Intracerebral Hematoma

Large traumatic intracerebral hematomas are uncommon; most are associated with extensive cortical contusions. They can be characterized as contusions in which larger, deeper vessels have been disrupted. Smaller, single hematomas that are not associated with contusion probably occur because of stress concentrations that result from impact or because of acceleration-induced tissue strains deep within the brain. Intracerebral hematomas have been poorly studied but may represent a form of tissue tear hemorrhage that accompanies diffuse axonal injury.

Tissue Tear Hemorrhages

Tissue tear hemorrhages are multiple areas of damage to blood vessels and axons occurring in combination with diffuse axonal injury. As such, these hemorrhages are caused by inertial head motion effects and therefore are not related to contact phenomena. They are distinct from the intracerebral hematomas described previously and are actually a part of the pathologic scenario of a severe form of diffuse axonal injury that results in immediate prolonged coma. Tissue tear hemorrhages are typically numerous, small, and located parasagittally and in the central portion of the brain.

Tissue tear hemorrhages appear in localized areas in which the brain's tolerance to shear forces has been exceeded, which

allows the tissue to separate sufficiently to tear both axons and small vessels. Their locations are characteristically in the superior medial frontoparietal white matter, corpus callosum, centrum semiovale, periventricular white and gray matter, internal capsule, and basal ganglia. In the brainstem, they appear in the dorsal area of the midbrain in the upper pons. Several factors contribute to these multiple foci of damage, including the presence of intracranial partitioning membranes, the geometric irregularities of the skull, and the plane of motion experienced by the head. Tissue tear hemorrhages represent areas of maximum acceleration-induced brain damage.

Subdural Hematoma

Three varieties of acute subdural hematoma manifest clinically. The first type of subdural hematoma is the most common form of vascular disruption; it involves tearing of parasagittal bridging veins located along the superior margin of the brain. It results entirely from inertial, not contact, forces. Because of their superficial location, parasagittal bridging veins are susceptible to damage during brief angular acceleration of the head. Common situations in which this acceleration condition occurs include falls, when the head strikes a broad, hard surface, and in cases of assaults in which a majority of the impact energy is used to set the head in motion. Under these loading conditions, differential motion between the brain and dura cause concentrated shear strain fields along the outer margins where the parasagittal bridging veins reside. The other two types of subdural hematoma are associated with contusion and laceration and are sometimes called *complicated subdural hematomas*. These result from the contact or acceleration effects that caused the primary lesion. Across all these types, if vascular tolerances are surpassed, subdural bleeding occurs, and mass effect from the hematoma may cause brain compression. Because of its similar mechanism, subdural hematoma may coexist with underlying hemispheric brain damage, usually diffuse axonal injury (see next section). This explains the frequency of cases in which the subdural hematoma is small but the underlying brain damage is greater than expected.

Diffuse Brain Injury

Cerebral Concussion

Concussion is a transient, generally reversible neurological dysfunction caused by trauma. All gradations of concussion are produced entirely by inertial loading and not from contact phenomena effects. Indeed, concussion does not occur with purely translational motion of the head. However, like other injuries, concussion can be observed in combination with injuries arising from contact phenomena simply because the contact loading produces both contact effects and angular rotational acceleration of the head. Angular rotational head motion causes the deeper structures within the brain to deform and causes the classical widespread disruption of brain function that underlies concussion. For a concussive injury, most of the strain is insufficient to cause structural damage. Instead, the damage to the structures may be either partially or completely reversible, depending on the severity of inertial loading. The precise location of the functional derangement of concussion continues to be debated. It remains unsettled whether angular acceleration affects principally the brainstem, the cerebral hemispheres, or many regions.

Diffuse Axonal Injury

Axonal damage appears to be an important pathologic condition that produces prolonged traumatic coma in cases without mass lesions. Diffuse axonal injury is considered a more severe form of cerebral concussion and, like concussion, is caused only by angular rotational acceleration and not by contact phenomena (although, as described previously, head impact is typically the cause of angular acceleration). However, diffuse axonal injury nearly universally coincides with other forms of contact or inertial injuries. The amount and location of axonal damage as a consequence of rotational acceleration probably determines the severity (depth and duration) of injury and probably has a strong influence on the quality of recovery. Critical factors in estimating the amount and extent of axonal damage include the magnitude, duration, and onset rate of the angular acceleration, as well as the direction of motion. In particular, diffuse axonal injury is produced by acceleration loading that is longer than the loading duration that can cause acute subdural hematoma. Thus diffuse axonal injury is most likely to occur when the head is impulsively loaded (i.e., no head impact) or when the impact occurs with a relatively soft, broad object (e.g., dashboard/headrest in a motor vehicle accident or another player's head in a sports collision). As described previously, injurious impulsive loading injuries are not common clinically because of the necessary levels of rotational acceleration; instead, angular acceleration associated with head impact is the most frequent circumstance producing diffuse axonal injury. Indeed, evidence has suggested that to obtain the magnitude of rotational acceleration needed to produce diffuse axonal injury, the head must strike an object or surface; this requirement raises the likelihood that superimposed contact injuries are also present. Furthermore, although all of the injury mechanisms that cause subdural hematoma and diffuse axonal injury are closely related, there is a marked difference in the type of accident that produces each condition. For instance, nearly all cases of diffuse axonal injury, especially in it severe form, arise from situations in which acceleration is more than 5 milliseconds in duration, common in motor vehicle collisions. Conversely, most subdural hematomas occur because of falls or assaults in which the impact duration is extremely short and the angular acceleration is abrupt.

The direction that the head moves plays an important role in the amount and distribution of axonal damage in a given situation. For equivalent levels of angular acceleration, the brain is most vulnerable to axonal damage if it is moved laterally. The brain tolerates sagittal movement best, and horizontal motions are somewhere between lateral and sagittal movements. However, sagittal motions are the most effective in producing vascular injuries to the superior margin of the brain, inasmuch as the motion of the brain in this plane is not severely restricted by intracranial membranes. To this end, the full-blown picture of widely scattered damage to cerebral hemispheres and brainstem, along with tissue tear hemorrhages, occurs most probably because of the spatial changes in the strain pattern induced by the falx and tentorium during lateral motions. Furthermore, the gyral geometry of the cerebrum and brainstem plays an important role in the response of the brain to rotational motions. In response to lateral head motion, small centers of rotation may occur in the superior frontal and temporal lobes.

CONCLUSION

The complex clinical injury patterns observed in patients with head injuries can be attributed primarily to three phenomena: contact forces, inertial forces, and blast overpressure. These three phenomena may cause damage to the brain and skull by causing the local deformation, or strain, of the bony or soft tissue that can result in either functional or structural derangement. As the name implies, contact phenomena appear when the head strikes or is struck by an object. Contact phenomena include local deformations of the skull that result in local or remote compressive, shear, and tensile strains in the underlying skull and brain. In comparison, inertial forces are generated by head motions that

occur during the traumatic event: most commonly, rotational and angular motion of the head.

Typically, inertial (acceleration) loading occurs in concert with contact phenomena (e.g., the head is struck by or strikes a rigid object). Most commonly, these motions are described as acceleration or deceleration of the head as it is set into motion or is stopped from moving. Head accelerations or decelerations of the appropriate magnitude, rates of onset, and direction together contribute to the type and severity of inertial injuries. Although it is a common cause in clinical situations, impact is not needed to cause sufficient head motion to result in mild, concussive-type injuries. Acceleration-based injuries include concussion, diffuse injury without hematoma (diffuse axonal injury), and most acute subdural hematomas and contrecoup injuries. In real-world situations, it is probable that some form of head contact is necessary to achieve levels of rotational acceleration and velocity considered responsible for acute subdural hematoma and severe forms of diffuse axonal injury.

Blast-induced overpressure injuries are a mechanism of injury for which research is emerging, but the exact mechanism or mechanisms that cause brain damage from this loading are not clear. Although this mechanism of injury can also be combined with both contact and inertial loading in the military environment, there is a new awareness that blast-induced pressure loading alone can lead to changes within the brain parenchyma that are difficult to detect, and further study is required.

By using the guidelines presented in this chapter, the clinician can distinguish and explain the numerous head injuries observed clinically on the basis of fundamental mechanisms of injury: contact forces, inertial forces, and potentially forces associated with shock wave propagation. Although instances of pure contact or inertially induced injuries do occur, the overwhelming majority of patients with head injuries exhibit a select constellation of lesions described in this chapter. This mixture of injuries, of course, suggests that contact and inertial mechanisms often manifest simultaneously. In most trauma situations, however, one injury mechanism is predominant, and the major clinical injury can often be categorized into one or two of the principal types. Evolving secondary injuries such as inflammation, reactive gliosis, edema, metabolic deficits, increased reactive oxygen species, excitotoxicity, hypoxia, and altered cell signaling—although beyond the scope of this chapter—are important considerations for patient outcomes, including acute medical management, neurological consequences, and potential chronic neurodegenerative sequelae of traumatic brain injury.

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